ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CORROSIVE GASTRIC STRICTURES

Dissertation submitted to

THE TAMIL NADU

DR.M.G.R. MEDICAL UNIVERSITY

in partial fulfilment of the requirements for the

award of the degree of

M.Ch BRANCH – VI

SURGICAL GASTROENTEROLOGY AND PROCTOLOGY

THE TAMIL NADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

AUGUST 2013

CERTIFICATE

This is to certify that the dissertation titled **"ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CORROSIVE GASTRIC STRICTURES"** submitted by **Dr.S.RAVIKUMAR** appearing for **M.Ch. (Surgical Gastroenterology and Proctology)** degree examination in August 2013 is a bonafide record, of work done by him under my guidance and supervision in partial fulfilment of requirement of the Tamil Nadu Dr.M.G.R.Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

DR. S.M. CHANDRAMOHAN
M.S., M.Ch., FACS.
Professor and Head,
Department of Surgical Gastroenterology
Centre of Excellence for Upper GI Surgery,
Rajiv Gandhi Government General Hospital &
Madras Medical College, Chennai-3.

DEAN
Madras Medical College
Chennai

DECLARATION

I solemnly declare that this dissertation titled **"ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CORROSIVE GASTRIC STRICTURES"** was prepared by me in the Department of Surgical Gastroenterology and Proctology, Centre of Excellence for Upper Gastrointestinal Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of **Prof.S.M.Chandramohan,** M.Ch, FACS, Professor & Head of the Department of Surgical Gastroenterology and Proctology, Centre of Excellence for Upper Gastrointestinal Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to The Tamil Nadu Dr. MGR Medical University, Chennai in partial fulfilment of the university requirements for the award of the degree of M.Ch Surgical Gastroenterology and Proctology.

Place: Chennai **DR.S.RAVIKUMAR**

Date:

ACKNOWLEDGEMENT

First, I would like to thank my patients, who subjected themselves for my dissertation work.

I whole heartedly thank **The Dean**, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai for allowing me to avail the facilities needed for my dissertation work.

I would like to express my humble gratitude to **Prof.S.M.Chandramohan**, M.Ch, FACS, Professor and Head of the Department of Surgical Gastroenterology and Proctology, centre of excellence for upper GI surgery, for his expert guidance and help rendered for the conduct and completion of my dissertation work.

I would like to thankfully acknowledge **Prof.D.Kannan, M.Ch,** Professor in the Department of Surgical Gastroenterology and Proctology, centre of excellence for upper GI surgery, for his constant help and guidance in the dissertation work.

I would like to thankfully acknowledge **Prof.G.Monahoran, M.Ch,** Professor and Head of the Institute of Surgical Gastroenterology and Liver Transplantation, Stanley Medical College and Hospital, Chennai for his constant help and guidance in the dissertation work.

I sincerely thank our Assistant Professors **Dr.A.Amuthan, Dr.R.Prabhakaran, and Dr.A.Benet Duraisamy** in the Department of Surgical Gastroenterology and Proctology, centre of excellence for Upper GI surgery, for their continuous inspiration and support in carrying out my dissertation.

Last, but not the least, I thank my fellow past and present postgraduates who helped me in carrying out my work and preparing this dissertation.

CONTENTS

INTRODUCTION

Corrosive injuries to the stomach and oesophagus is one of the common condition in developing countries like India .It may result in major morbidity and mortality particularly in the younger age group. Corrosive poisons are those substances which corrode and destroy tissues through direct chemical action. They almost always act locally and have few systemic effects. Caustic poisonings can occur after ingestion of caustic substances, such as acids and alkalis.

The most commonly abused substance among acids is hydrochloric acid. Alkali ingestion is more common in the western developed countries. In India, acid ingestion is the cause of corrosive oesophageal strictures in about 75% of patients. Accidental ingestion of acids and alkalis has been reported to be less common in western countries at present as the effective publicity and the enforcement of the law to ban the sale of strong acids and alkalis in the open market. Accidental ingestion is more common in children and drunken adults, due to careless storing of chemicals .Ingestion with suicidal intent is more common in India due to free availability of the caustic agents mostly used for toilet cleaning purposes.

Post-corrosive injuries with different degrees of severity can occur along the entire oesophagus particularly in the middle and distal part, more precisely at the site of physiological narrowing. In the stomach,

they can appear in the antrum and pylorus[1,2]. Strictures isolated to the stomach due to acid or alkali is not an uncommon entity in our tertiary centre where lot of patients particularly corrosive injuries are referred.

The oesophageal and gastric involvement usually depends upon the nature of the corrosive ingestion, amount of ingestion and caustic concentration, length of contact and the swallowing act[3]. Acids usually damage the stomach and alkalis damage the esophagus. Acid injury to the stomach more commonly due to mucosal injury results in coagulation necrosis, transformation of proteins, and haemoglobin. It is usually due to prolonged duration of contact [4]. Although acids can reach quickly from the oesophagus to the stomach, they lodge in the pre pyloric region because of spasm of the pylorus .

The more the duration of contact within the prepyloric mucosa, the more the damage result in prepyloric stricture. Strictures may involve any part of the stomach including antrum, body, and the pyloroduodenal area or diffusely throughout stomach depending upon the volume of caustic ingested. Strictures may be isolated to the stomach or it may be associated with strictures of esophagus.

Although alkalis usually damage the esophagus severely, it can also damage the stomach[5]. Alkalis transform tissue proteins into proteinases and fats into soaps which result in liquefaction necrosis[6]. Since they are more viscous, they attach to the oesophageal mucosa with

only small amount may reach to the stomach. Severe acute injuries are usually lethal and are most often due to tracheal necrosis, perforation of the oesophagus and stomach, and the consequential mediastinitis and peritonitis[7]. Caustic substances, which have a pH of less than two or greater than twelve are more dangerous and may result in severe damage of tissues.

Acute corrosive injuries can result in particular histological appearance of the oesophageal and gastric walls where thrombosis of the small vessels can be predominantly seen. Temperature may be high in the cells due to metabolic changes, and after a few days, they are overtaken by bacterial invasion[8]. The healing process usually begins in three weeks after ingestion by collagen deposition in the first week, later with development of granulation tissue and fibrosis, which leads to the incidence of oesophageal, stomach strictures during the next eight weeks to eight months[9]. Oesophageal stricture most often develops at the cricopharyngeal region, at the site of the aortic arch, tracheal bifurcation and the region of lower oesophageal sphincter. Narrowing in the stomach mostly occurs in antrum and the pylorus. Studies indicate that clinical signs are not always helpful in predicting the degree of injury and subsequent stricture formation. Esophagogastroduodenoscopy (EGD) is the most effective method for establishing the severity of injury and treatment planning.

The injury encountered at a major hospital does not reflect the overall data since patients with high grade caustic gastric and oesophageal injuries may die at peripheral hospitals. The spectrum of gastric injury due to corrosives can vary from acute partial or total gastric mucosal or transmural necrosis to chronic gastric injuries of different types. Gastric lavage by the administration of active coal is contraindicated in the acute settings since second exposure of the oesophagus to the caustic agent, causing extra damage.

Antibiotics, cortico-steroids, nutritional support, endoscopic dilatation and stenting and surgical treatment, are well described modalities in the management for corrosive injuries

Response to balloon dilation is not good for corrosive gastric stricture compared to response rate that occur for peptic strictures[10]. Corrosive gastric strictures may need frequent dilations than peptic ulcer-related strictures [11]. Strictures occurring at the distal gastric site may need sufficient dilatation to prevent obstructive symptoms, but at the cost of increased risk of perforation. Resistance to initial balloon dilation may reflect worse long-term results. Surgical correction has been recommended for these patients[12]. Long segment, tortuous, angulated or oedematous benign gastric outlet strictures poorly respond to balloon dilation[13]

Electrosurgical incision and intra lesional steroid injections may be useful for augmentation of the effect of balloon dilation. [14, 15, 16]

Endoscopic stenting has been established as an effective way to treat strictures due to corrosive agents [17.] Surgical management depends upon the extent of gastric involvement and presence of associated esophageal stricture.[18]

Strictures produced by corrosive injury are a challenging problem to both surgeon and the patient. Many studies were available in the management of corrosive esophageal strictures. But, only few data are available in the world literature regarding the incidence and management of corrosive gastric strictures. As ours is a high volume surgical centre for the management of corrosive injuries in India, we get all varying patterns of presentation of gastric strictures. This study analyses all patients admitted in THE DEPARTMENT OF SURGICAL GASTROENTEROLOGY, CENTER OF EXCELLENCE FOR UPPER GASTROINTESTINAL SURGERY, RAJIVGANDHI GOVERNMENT GENERAL HOSPITAL for the management of corrosive gastric stricture between August 2010 to February 2013.

From this study, we describe our experience in managing corrosive gastric strictures and we have formulated a new working Classification in managing corrosive gastric stricture. It will help in deciding the surgical strategy to be adopted in the management of gastric stricture whether isolated to stomach or combined with esophageal stricture.

AIM OF THE STUDY

To find out the mean duration between corrosive consumption and development of the gastric stricture

To find out the common site of stricture in the stomach

Whether isolated or combined with esophageal stricture

Type of surgical procedure in relation to site of stricture and outcome after the procedure and to formulate a new working Classification which will help in deciding the type of surgery in patients with corrosive gastric stricture

REVIEW OF LITERATURE

The estimated prevalence of corrosive poisoning is 2.5-5%.The morbidity is above 50% and the mortality is 13% [19].Corrosive ingestion is a common cause of upper gastrointestinal tract benign stricture in India [20].Corrosive poisons are those substances, which corrode (to eat away) and destroy tissues through direct chemical action.

The common caustic agents include:

Strong acids and alkalis

Concentrated weak acids and alkalis

Oxidizers (with neutral pH)

Alkylating agents

Dehydrating agents

Halogens and organic halides

Phenol

Strong acids

Inorganic acids (mineral acids)

1. Sulphuric (Car battery fluid)
2. Nitric (Metal cleaners)
3. Hydrochloric (Descalers, metal/toilet bowl cleaner)
4. Hydrofluoric acids (Rust removers)

Organic acids

Carbolic, oxalic and salicylic acids - These are weaker in action compared to inorganic acids and are usually absorbed into circulation promoting local and remote action

Strong alkalis

Anhydrous ammonia

Sodium hydroxide

Potassium hydroxide

Ammonium carbonate

Sodium carbonate

Potassium carbonate

The most commonly ingested substance among acids is hydrochloric acid. It is often used in India compared to USA where its abuse is less than 5%[21] . In India, toilet cleaning hydrochloric acid is easily available and is the most common caustic consumed by poor people. The next most frequently consumed agent in India is gold solvent, which is a combination of nitric acid and hydrochloric (1:3) ratio [22].

Factors Determining Corrosiveness

Factors that determine corrosiveness include:

Physical form: Solid/liquid

Duration of contact with tissue

Concentration of agent

Quantity of agent

pH of agent: pH <2 and >12 are more corrosive

Food: Presence or absence of food in stomach

Acids which are titratable or reserve of alkali (TAR): It estimates the requirement of neutralizing substance to lower the pH of a caustic agent to physiological pH of the tissue [23]

Mechanism of Action of Corrosive Agents

Alkali ingestion: Causes liquefaction necrosis.

It includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, sub mucosal vascular thrombosis and cell death[24]

Acid ingestion: Causes coagulation necrosis.

It includes coagulation of cellular proteins, and its precipitation. It in turn results in conversion of haemoglobin to haematin. In this process, hydrogen ions desiccate epithelial cells resulting in eschar formation. This process leads to erythema, edema, sloughing of mucus membranes, ulceration and necrosis of tissues [25]

Both acids and alkalis cause fibrosis and cicatrisation (stricture formation), Squamous epithelium of oesophagus is more resistant to acid than columnar epithelium of stomach. On the contrary, Squamous epithelium of oesophagus is more sensitive to alkali than columnar epithelium of the stomach. Hence oesophageal strictures are more common in alkali poisoning and gastric strictures are more common in acid poisoning.

Pathologic classification of corrosive injuries

It is same like the classification of skin burns due to thermal injuries.

First degree: Injury is superficial in nature, followed by oedema and erythema of mucous layer. Regeneration of the damaged mucous layer takes place within a few days and does not produce damage like stricture and scar formation.

Second degree: Corrosive penetrates through the sub mucosal layer into deep muscular layer of the involved areas. After 1 to 2 weeks, it results in deep ulcerations and granulation tissue in the oesophagus and stomach. It produces fibroblast reaction and collagen tissue .Soon after it will lose its humidity and is subjected to contraction over a period of several weeks to months. These events along with the nearby injuries can result in narrowing of oesophageal or stomach lumen within the next eight weeks to eight months and may produce stricture or stenosis

Third degree: Oesophageal or stomach perforation

Consequences of corrosive Injury

Corrosive injury may lead to the following [26]

Necrosis: Usually results within seconds of exposure to corrosive agent

Ulceration and perforation: Within 24-72 hours of exposure

Fibrosis: Within 14-21 days of exposure

Stricture: Weeks to years after the exposure

Malignancy: May develop after decades of alkali exposure.

Malignancy may develop after many years in the damaged esophagus or stomach although absolute risk is very small. The risk about

developing gastric malignancy is not well known. Grey first described findings of squamous metaplasia of stomach in a patient who had consumed acid.O D'onnal and Eaton confirmed the same risk [27]. Since strictured stomach may lead to protein losing gastropathy, resectional surgeries for cicatrized stomach rather than bypass is preferred by some surgeons.

Clinical features

Clinical presentation depends upon timing of presentation after consumption of corrosives

Acute caustic intoxications – usually present with constant burning pain in the mouth, throat, and chest and severe pain in the abdomen followed by nausea and vomiting, hyper salivation, and difficulty in swallowing. Symptoms usually appear immediately after caustic ingestion.

Usual findings on clinical examination are ulcers or whitish plagues in the oral cavity, palatal mucosa, and pharynx. Patients can also present with dyspnoea, tachypnea, dysphonia, and aphonia due to laryngospasm .Most severe injuries are those with endotracheal or bronchial necrosis with mediastinitis, due to aspiration of the corrosive substance.

Absence of oropharyngeal injuries does not rule out severe injuries of the lower segments. One survey showed about 37% of presence of oesophageal injuries (II and III degree) among patients that had not developed evident oropharyngeal damages. By contrast, some reviews reported that seventy percent of individuals with severe oropharyngeal injuries did not have noticeable esophageal damages. Therefore, injuries of the oropharynx are not a reliable indicator for the eventual burns located in the oesophagus or stomach [28].

Corrosive injury can involve in pharynx in 10% of the patients. Oesophagus can be involved in 70% patients .Middle segment of oesophagus is affected more commonly than upper and lower segment of the oesophagus. Middle segment is involved in about 65% of cases compared to upper segment (15%) and lower segment (20%).Entire oesophagus can be affected in 18% of the patients. Stomach involvement is in 20% of cases.Antrum is affected in 91%of patients when the stomach is involved. Whole stomach is involved in 90% of patients [29]

Oesophageal or gastric perforation may be the most serious complication in the acute phase. Post-corrosive injuries usually appear in the first two days or they may be delayed up to two weeks after caustic ingestion [30].

Chronic gastric injuries - usually present with abdominal fullness, abdominal discomfort, vomiting or early satiety. It occurs less commonly

than oesophageal stricture which may occur five to six weeks after the consumption. It can even occur after years [31] of injury but the concomitant oesophageal stricture may mask an underlying gastric injury.

Gastric malignancy is a rare and long term insult following caustic ingestion [32]

Investigations

Plain X ray chest and abdomen:

1. Useful in acute presentation to rule out oesophageal or gastric perforation, by mediastinal air or air under the diaphragm respectively

2. It is useful in gastric stricture patients to find out aspiration and gastric dilatation in case of double stricture (gastrocele)

3. In patients with absolute dysphagia, where endoscopy or barium swallow is not very much useful

Esophagogastroduodenoscopy:

The most optimal time for urgent scopy is in the first 12-24 hours after ingestion of the caustic substance. This diagnostic procedure should be avoided between the 4th and the 14th days after ingestion because of

intense inflammatory changes, vascular thrombosis and healing process of the post corrosive lesions [33].

It is useful for

1. To assess the mucosa
2. The site and level of narrowing and number of strictures
3. Strictures- dilatable/ non dilatable
4. Stomach capacity
5. Evidence of gastric outlet obstruction, status of the antrum and duodenum

Endoscopic grade of caustic injuries in the upper gastrointestinal tract is helpful in diagnosis and treatment of acute caustic intoxications. It has a role to identify the patients who will be prone for stricture.

Kikendall has classified corrosive injuries by endoscopic appearance.

Grade – I injuries include edema and erythema of the mucosal membrane

Grade – II injury include ulcers which is superficial, mucosal erosions haemorrhage and blisters

Grade - II b injuries include circumferential involvement

Grade – III injuries include ulcers, grey or brownish-black in colour

Grade – IV injuries include perforation.

Zargar has also classified corrosive injuries by endoscopic appearance. His classification is widely followed.

Grade 0 - represents normal appearing mucosa on endoscopy

Grade I - mucosa appears to be oedematous and erythematous

Grade II A - includes superficial ulcers, erosions, haemorrhage and blisters

Grade II B - includes circumferential involvement

Grade III A - include ulcers which are in brownish-black or gray or in colour, focal involvement

Grade III B - include ulcers which are brownish-black or gray in colour, but extensively involved

Grade IV - Oesophageal or gastric damage resulting in perforation

The course of injury and complications are predicted by the grade of injuries.

Grade I - these type of patients don’t have the oesophageal stricture risk

Grade IIB - these patients have a chance of 75% stricture risk.

Grade III - All patients (100%) have a chance to develop stricture

< Grade IIB - Malignancy risk is not increased compared to the general population

> Grade IIB - Malignancy risk is 1000 (latency 20-40 years) times compared to the general population.

Grade II and III - these type of patients have increased risk of complications that include stricture, tracheo-oesophageal fistula, altered motility, erosion into major vessels

Barium swallow

Although it is not helpful in acute injuries, it is useful in chronic injuries

1. To identify the site of oesophageal stricture ,length of the stricture, number of strictures and type of gastric stricture
2. To assess the distal flow
3. To identify the stomach capacity
4. To identify the deformity and evidence of gastric outlet obstruction

Endoscopic ultrasound and computerized tomography

Useful to assess the depth of the corrosive injuries more precisely than endoscopy alone

Meckel's scan

It is also helpful to assess the severity of injury in the stomach [34]

Indirect Laryngoscopy and Bronchoscopy

Helpful to assess the vocal cord status and respiratory passage

Malnourished patients should undergo nutritional evaluation

Surgical candidates -will have to undergo cardiac and respiratory evaluation other than renal and hepatic function

Management

Management of corrosive injuries depend upon the time of presentation after the consumption of corrosives

The aim of the therapy in the immediate management after corrosive ingestion is to prevent complications like perforation and to avoid progressive fibrosis and stenosis of the stomach. Emergency surgery is needed in cases perforation of the stomach though it may be difficult to predict it at the initial stages.

Neutralization of corrosive substances

General teaching in the management of corrosive poisoning is avoidance of neutralization. To be effective, it should be done within hour after ingestion of a corrosive agent [35]. Alkalis need neutralization with vinegar, lemon or orange juice. Acids need neutralization with eggs, milk or antacids. Caution is needed in using sodium bicarbonate, since it will produce carbon dioxide, which may increase the complication like perforation.

Emetics should not be given as re-exposition to caustic agent can lead in to exacerbation of injury. Active coal is also contraindicated.

Corticosteroids

Although role of systemic steroids to prevent stricture formation is controversial, several studies showed that corticosteroids had no significant influence on prevention of post-corrosive stricture in acute corrosive injury settings[36]. Some studies showed the usefulness of intralesional steroids in corrosive pyloric strictures [37].

Antibiotics

Still now, controlled studies are not available to confirm the need of antibiotic administration although animal studies suggested the role of bacteria in the formation of fibrosis after injury[38]

Nasogastric tube placement

Blind Nasogastric tube placement can produce oesophageal perforation. Early placement of a Nasogastric tube may be desirable particularly in high-grade injuries to allow access to the stomach. In high grade injuries (grade IIB or III), feeding tube should be placed under endoscopic guidance to maintain access to the stomach.

Nutrition

Corrosive stricture patients won't able to take per orally due to severe damage of the gastro intestinal tract. These patient's nutritional status can be deranged within a short time period due to hyper catabolic and negative nitrogen balance. Nutrition is lifesaving particularly in patients who cannot take anything by mouth to avoid the risk of malnutrition [39].

Proved effects of artificial feeding in patients with caustic strictures include

- Infections rate will be reduced
- Low risk of aspiration pneumonia
- Risk for pulmonary embolism may be reduced

The need and artificial nutrition type depends upon the damage of oesophageal or gastric involvement visible by upper GI scopy

I and II A degree of damage – Need total parenteral nutrition during the first 24–48 hours. Liquids can be started after that and continued until the 10th day. Food intake can be started after tenth day.

II B and III degree of damage –Oral fluids and solid diet must not be allowed in the early period. During this time, the patient can be fed by nasogastric tube or naso jejunal tube, gastrostomy or jejunostomy and parenteral route by peripheral or central vein. It is because swallowed particles may enter the granulocytes of the oesophageal wall and increase the risk of inflammatory insult [40]. Oesophageal rest may be needed up to tenth day after corrosive ingestion .But it may be delayed until the 15th day as recommended by some authors [41].

There is no uniform consensus regarding when to start oral diet although some recommend taking only liquid diet up to 48 hours after ingestion if the patient can swallow saliva [42].

Endoscopic management

Although endoscopic dilatation is well documented in managing oesophageal stricture, its role in the management of gastric stricture is not well documented. Only thirty five percentages of patients with corrosive strictures had response to balloon dilation, compared with response rate of seventy percent for the patients with peptic strictures [43]. Corrosive

stricture patients need more dilations than those with peptic ulcer-related strictures [44].

Recent report has shown repeated balloon dilations (approximately 6 sessions) resulted in achieving long-term resolution (symptom free for 18 to 58 months) in 95% (39 of 41) of the patients with short-segment (<2.5 cm), corrosive-induced gastric outlet strictures [45].

Gastric outlet strictures need sufficient dilatation to relieve symptoms of obstruction, but at the cost of increased risk of perforation. It is necessary to decide the optimal diameter of dilation. The optimal diameter may vary with the type of stricture (anastomotic vs. non-anastomotic stricture). 20 mm diameter may prevent symptoms of obstruction with no major complications in anastomotic strictures[46]. 15 mm diameter may be sufficient to relieve symptoms of obstruction[47] for non-anastomotic strictures.

Angulated, tortuous or oedematous benign gastric outlet strictures are usually refractory to balloon dilation.

Role of stenting

Stents should be used selectively in patients with resistant benign gastric outlet strictures as it has the disadvantage of high risk of migration.

Intralesional steroid injections

Singh K et al described the usefulness of intralesional steroid injections to augment the effect of balloon dilation in 3 patients with caustic induced pyloric obstruction [48].

Surgical management

Surgery remains the main modality in managing corrosive gastric stricture[49].

The choice of ideal procedure is determined by

1. Site of stricture in the stomach

2. Extent of Stricture

3. Concomitant involvement of oesophagus

4. General condition of the patient

The appropriate time for surgical management for a chronic corrosive gastric injury is debatable issue[50]. Some authors recommend early surgery to these types of patients [51]

Whenever the surgery is planned, patient's nutritional status must be stabilised by paraenteral nutrition or whenever possible by enteral nutrition either by nasoenteral feeding or by feeding jejunostomy.

Different Surgical methods were described by various authors to manage corrosive gastric strictures in the literature.

Removal of cicatrized portion of stomach may obviate the chance of malignancy. The incidence of gastric malignancy is not well known. Grey first described in 1948, findings of squamous metaplasia in the stomach of a patient who had consumed acid .O'Donnell confirmed it [52].

Gastric resection either in the form of distal gastrectomy for antro pyloric stricture or subtotal /total gastrectomy for more extensive stricture was recommended by Shaleen agarwl group [53].In their experience resection surgery was safe and free of both short term and long term complication.

Ananthakrishnan et all has classified chronic corrosive gastric injuries into five types based on the location and extent of gastric stricture

Type 1: Stricture in the prepyloric region

Type 2: Extension of the stricture up to antral region

Type 3: Stricture in the mid body of the stomach

Type 4: Diffuse involvement of the stomach

Type 5: Gastric and duodenal stricture

Management according to Ananthakrishnan et all

Type 1: It can be managed by limited resection and Billroth I anastomosis

Type 2: He advocate anti colic loop Gastrojejunostomy in this type of patients. Since the strictures are long, gastro duodenal anastomosis is difficult and not possible. He also recommended to avoid retro colic route for Gastrojejunostomy as it might disturb the arcade of middle colic vessels and also make the mobilization of the colon at later period for oesophageal bypass more difficult.

Type 3: These types of patients need distal gastrectomy.

Type 4: When only the stomach is involved and also well preserved general condition, they are managed by a total gastrectomy. In case of associated oesophageal injuries, they usually present with poor general condition. They are treated by colonic bypass for the oesophagus and the distal part of the colon is anastomosed with the proximal jejunum, leaving the stomach undisturbed.

Type 5: These patients are treated by anti-colic Gastrojejunostomy since these patients will not able to withstand duodenal resection.

Kaushik R et all recommended that vagotomy is not needed for those patients with antral stricture associated with gastric outlet

obstruction while performing Gastrojejunostomy. Since the stomach's capacity of secretion is deranged after ingestion of caustic substances, the patients undergo ''physiological antrectomy'' [54].

In case of combined stricture of the oesophagus and stomach, they can be managed at the same sitting. Double stricture can be managed with Antrectomy with coloplasty, Gastrojejunostomy with coloplasty or antrectomy with retrograde dilatation. Definitive reconstruction can be done as staged procedure particularly in patients who cannot tolerate major resection at the same sitting [55].

In case of precedence of the gastric stricture to oesophageal stricture, it can be managed with a limited gastric resection and reconstruction, leaving oesophageal bypass at later date if necessary.

It can also be managed by feeding jejunostomy to improve the nutrition, and gastric or combined gastro oesophageal reconstruction on a later date. But gastric strictures can present even weeks to months after esophageal obstruction.

Delayed obstruction of gastric outlet can be a big problem if an oesophageal bypass has been performed. It can present with dilatation of the conduit[56]. This condition should not be misdiagnosed as colo gastric stenosis. Diagnosis can be readily identified by endoscopy, and the

stenosis may be treated with either distal gastric resection or gastrojejunostomy distal to the cologastric anastomosis.

The situation is more troublesome if delayed gastric outlet obstruction manifests after a gastric pull-up for esophageal bypass after corrosive injury. In those cases, gastric resection or gastroenterostomy is not possible, and the surgeon will have the only option of jejunal interposition.Pyloroplasty may also be helpful in rare instances.

MATERIALS AND METHODS

Patients admitted for corrosive gastric stricture management in **Department of Surgical Gastroenterology, Center of Excellence in Upper G.I Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai – 3** during the period from August 2010 to February 2013 were analyzed.

All patients admitted in our department for the management of corrosive stricture between August 2010 to February 2013 were analyzed

Inclusion criteria:

Patients with isolated corrosive gastric stricture

Patients with gastric stricture extending into duodenum

Patients with combined esophageal and gastric stricture

Exclusion criteria:

Patients with acute corrosive injury

Patients who required emergency surgery for complications

Isolated esophageal stricture

Following data were collected and specifically analyzed

Age group

Sex

Occupation

Nature of ingestion

Type of corrosive ingested

Duration between ingestion and development of stricture

Location of corrosive stricture in the stomach

Association with esophageal stricture

Type of surgeries performed

Short term outcome after surgery

Clinical data including history of difficulty in swallowing, cough with expectoration, difficulty in breathing, abdominal pain, abdominal distension, vomiting & fever were collected. History of alcohol intake including amount, frequency and associated comorbid illness like Diabetes Mellitus, Hypertension, and Bronchial Asthma & Tuberculosis were also noted.

Patient's general examination, ENT examination and psychiatric opinion were analyzed

All basic biochemical investigations including Complete blood count, Serum electrolytes, Blood sugar, Renal function tests, Urea, Serum Creatinine, Liver function tests including bilirubin, SGOT, SGPT, SAP, Serum albumin and Prothrombin time were noted.

Specific investigations like

Barium swallow

To locate the stricture site in the stomach and coexistent esophageal stricture

To assess the distal flow

To assess the stomach capacity and evidence of gastric outlet obstruction

Upper gastro intestinal copy

To assess the mucosa, site of narrowing and number of stricture

To look for any evidence of gastric outlet obstruction

To look for the status of antrum and duodenum

USG abdomen

To look for gastrocele

CECT Chest & Abdomen

In selected patients who were unable to swallow & to rule out complications

Barium enema and colonoscopy

To assess the colon in patients planned for oesophageal substitutes

Preoperative performance status, cardiorespiratory status, hydration status & nutrition status were assesed. All patients were encouraged to have incentive spirometry at least 2 weeks prior to surgery. Informed consent was obtained from all the patients explaining the condition and magnitude of morbidity and mortality after surgery.

Intra operative findings of corrosive stricture and nature of surgery performed were analyzed.

Short term post op complications including the following are noted

Respiratory infection

Wound infection

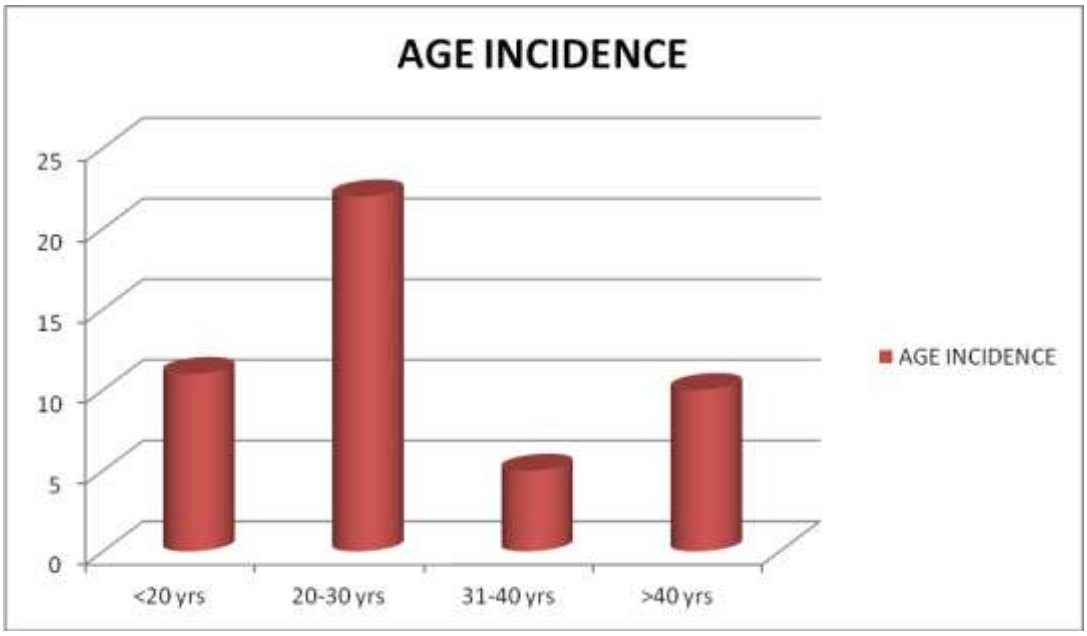
Anastomotic leak

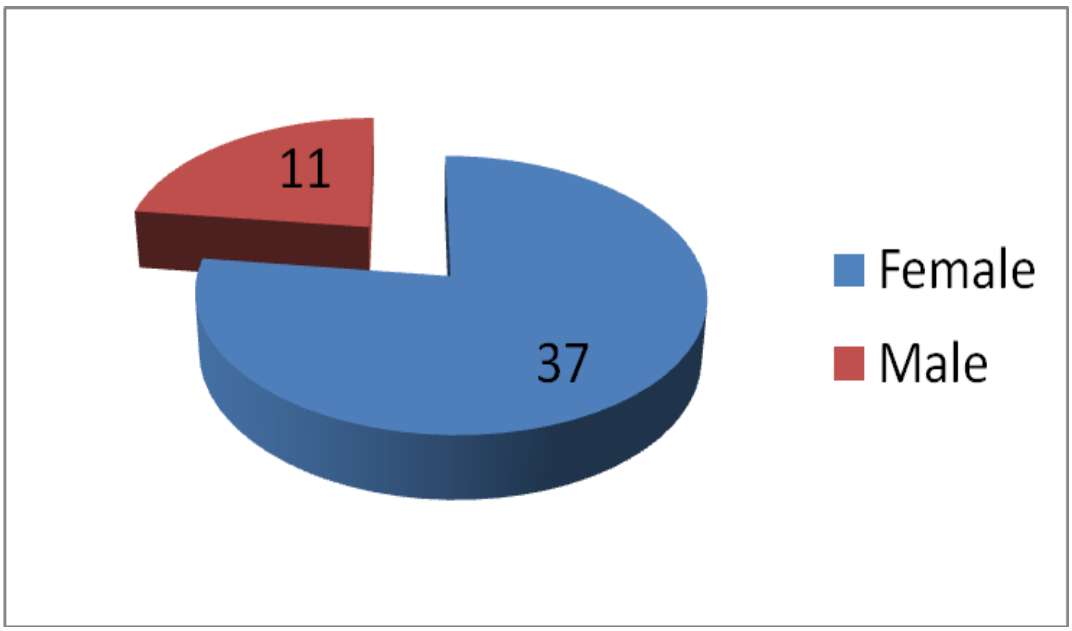
Cause of mortality

Length of hospital stay

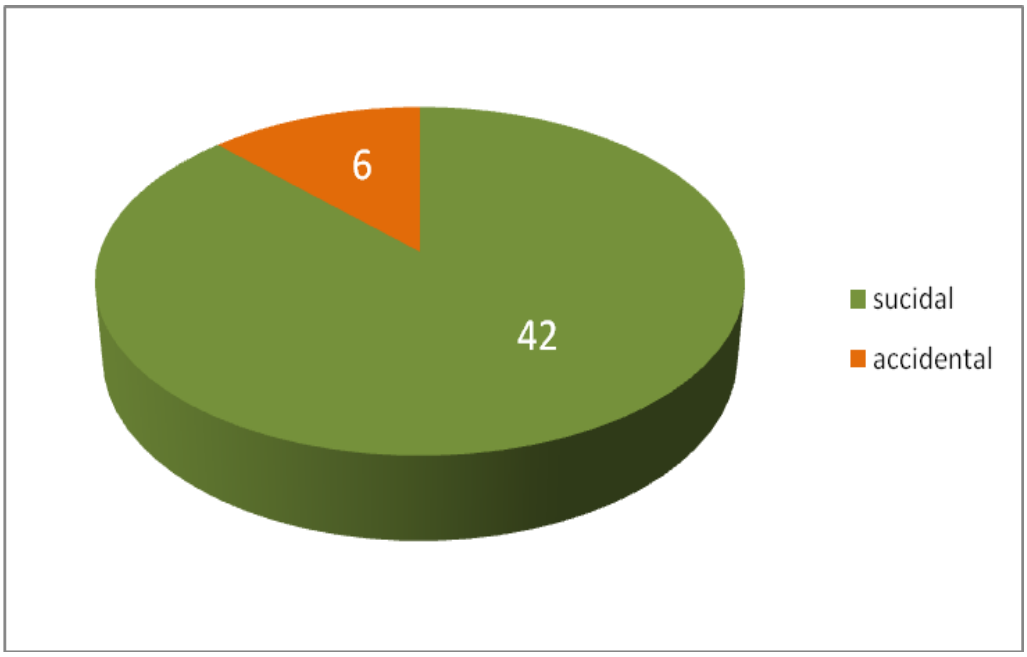
RESULTS

Among 48 patients included in the study, the age group commonly affected was between 20-30 years and commonly affected sex were females (F/M-3.36:1) as shown in figure 1 & 2





Acid ingestion was more common than alkali ingestion in our series. Among acids, toilet cleaning acid which contains hydrochloric acid was the most common acid ingested by the patients. Most of the patients ingested acids by suicidal intention.



All the patients had undergone hospital admission during the acute phase in medicine department in our hospital or elsewhere.

They presented to our department with

Complaints	Patients
Dysphagia	21
Respiratory tract infection	20
Dull abdominal pain	14
Acute abdominal pain and respiratory embarrassment	2
Vomiting	32
Weight loss	48
No abdominal symptoms	33

The time interval between corrosive ingestion and admission varied from patient to patient. (2 months to 35 months)

All patients underwent routine hemogram to check for haemoglobin levels, renal function test, serum electrolytes, liver function test to look for albumin level, chest x ray to look for cardiopulmonary status, and USG Abdomen.

Among 48 patients, 13 patients (27.08%) had hyponatremia (sodium <130).9 patients with type I stricture, 3 patients with type VII

stricture and 1 patient with type IV had hyponatremia. Hyponatremia was corrected prior to surgery by appropriate fluid management.

15 patients (31.25%) had lower potassium level at the time of admission.8 patients with type I stricture, 3 with type VII, 1 each with type II a, type II b, type III, type IV had hypokalaemia. Hypokalaemia was corrected by appropriate fluids.

10 patients (20.83%) had hypoalbumnemia (albumin <3.5). Hypoalbuminemia was corrected by giving intravenous albumin solution.

Barium meal study was done for all of these patients to locate the level of stricture in the stomach, associated stricture in the oesophagus, and coexisting trachea-oesophageal fistula. Upper gastro intestinal scopy was done prior to surgery to confirm the barium swallow findings. Among 48 patients, stricture was isolated to the stomach in 28 patients .Associated oesophageal stricture was noted in 20 patients.

Otorhinolaryngologist's opinion and physchatric opinion were obtained routinely for all patients. Barium enema and colonoscopy were done to assess the colon particularly in patients planned for oesophageal substitutes. We don't do mesenteric angiography routinely in all cases.

Surgical management was decided by based on barium meal, Upper gastro intestinal scopy and lastly by intra-operative findings.

Surgery was decided according to the level of stricture. Our working classification of corrosive gastric strictures into seven types is based on :

The site of stricture in the stomach

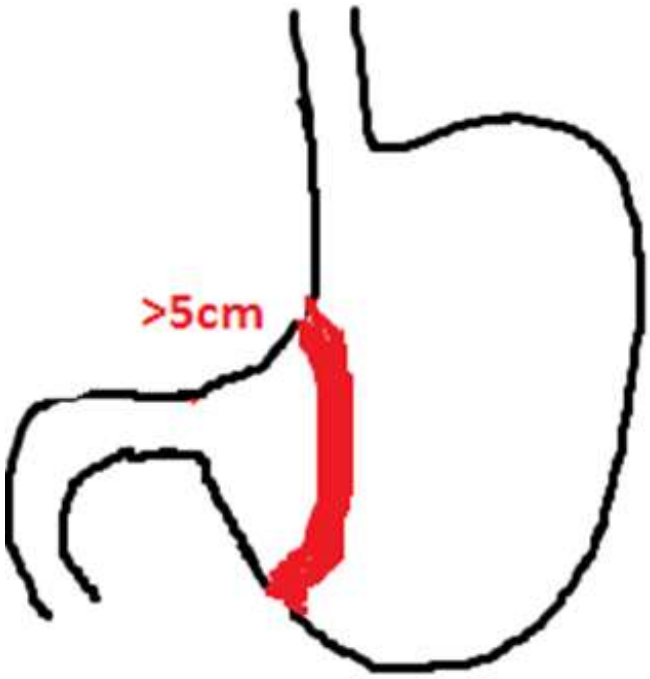
The extent of the stricture

Associated stricture of the oesophagus

According to that,

Type I – Stricture < 5 cm from pyloroduodenal ring

Type II – Stricture > 5 cm from pyloroduodenal ring



Type II A – Stricture < 1cm, Type II B – Stricture > 1cm

Type III – Mid body stricture

Type IV – Diffuse Gastric Stricture

Type V – Proximal or Gastroesophageal junction Stricture

Type VI – Distal gastric stricture involving duodenum

Type VII – Double Stricture (Stricture both in the distal stomach and Gastroesophageal junction Stricture)

Peroperative assessment includes;

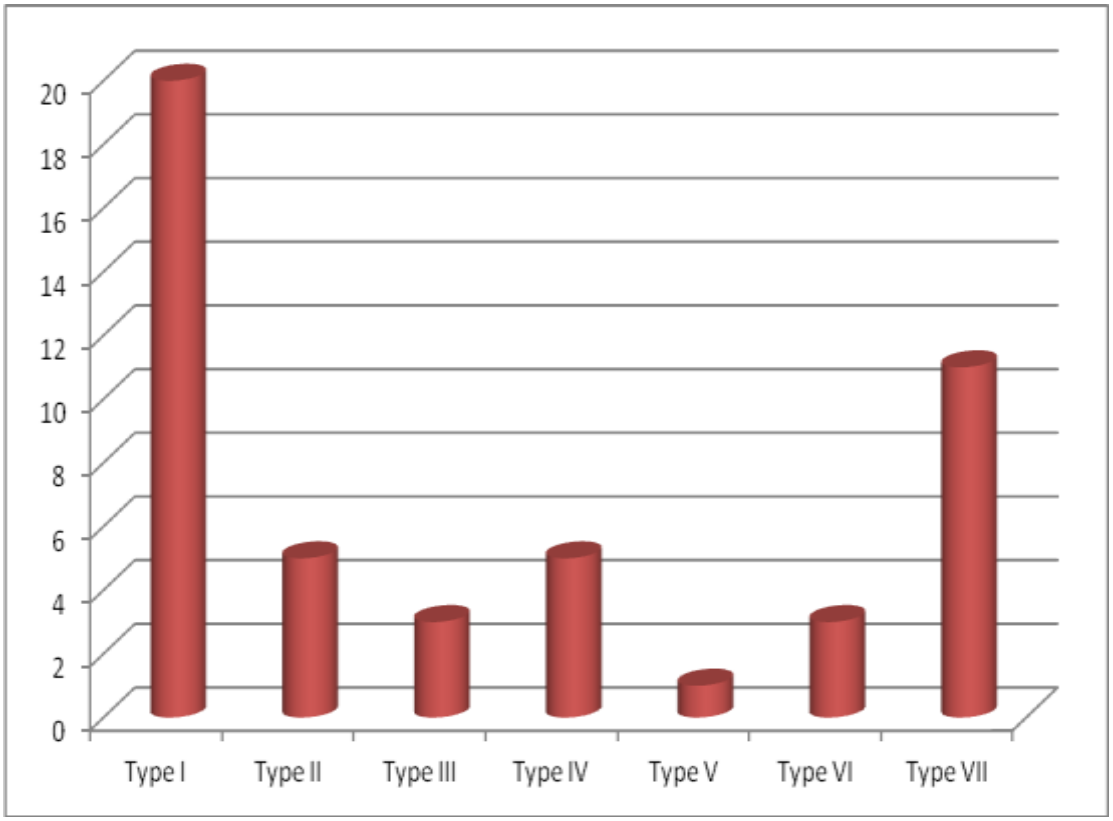
Assessing the site of the stricture

Oedema

Extent to the duodenum and oesophagus

We prefer to do resection rather than bypass of antral stricture.

Among seven types, type I stricture (41.66%) was the most common and type VII stricture (22.91%) was the next most common as seen in the following figure



We had 20 patients with type I corrosive gastric stricture

These patients were managed by

Arcade preserving antrectomy

Conventional antrectomy

Arcade preserving antrectomy was done in 12 patients.We used this arcade preserving technique whenever possible.This can be helpful in future for these patients to use stomach as conduit for management of esophageal stricture,if colon is unavailable.

Steps in arcade preserving antrectomy include

Duodenal kocherisation

Dissection close to greater and lesser curvature of the stomach and first part of duodenum with care to preserve right gastric vessels and right gastroepiploic vessels.

Duodenum transected by using stapler or knife.

Stomach transected by using knife or staper.

Reconstruction done by either Bilroth I or Bilroth II anastamosis.

We often do totally stapled Bilroth I anastamosis for type 1 corrosive gastric stricture. (10 out of 20 patients)

Steps of totally stapled bilroth anastamosis include

Arcade of stomach is preserved as stated above

Gastrotomy is made at the level of th stricture

Anvil of SDH 25 is introduced through the gastrotomy site and pushed to the first part of the duodenum

Duodenum is transected distal to the pylorus by using TLC 75 blue stapler

Anvil is brought through close to staple line

Trocar of the stapler is introduced through the gastrotomy site and brought to the posterior wall of the stomach

Trocar of the stpler SDH 25 is connected to the anvil

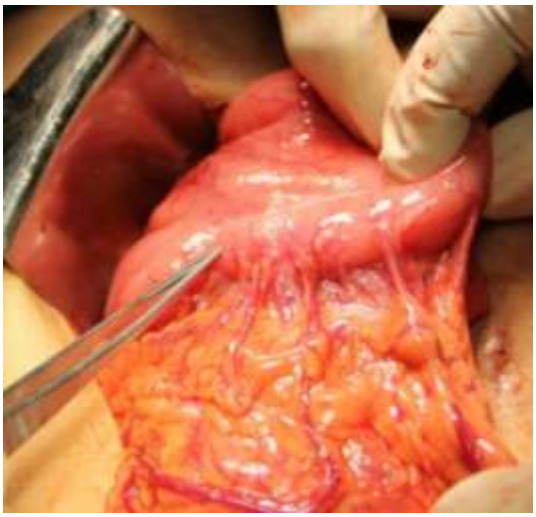
Firing of the SDH 25 stapler is done after the firing range noted in the stapler

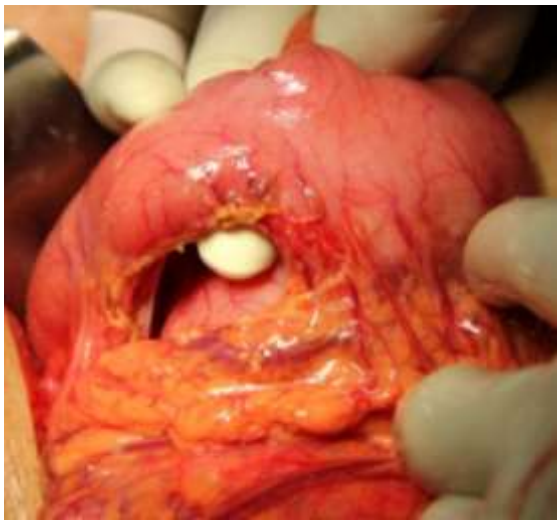
Side to end gastroduodenal anastamosis was completed

Finally, with the help of TLC 75, gastric transection done, specimen sent for biopsy.

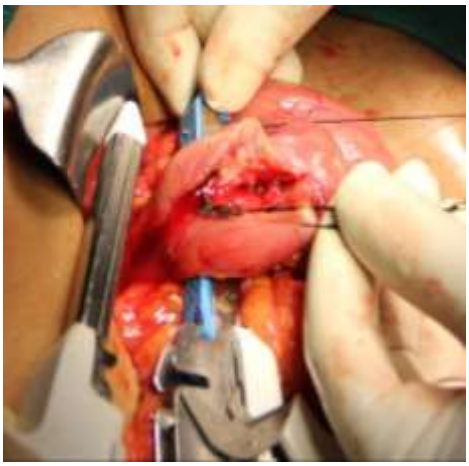
BARIUM SWALLOW – TYPE I

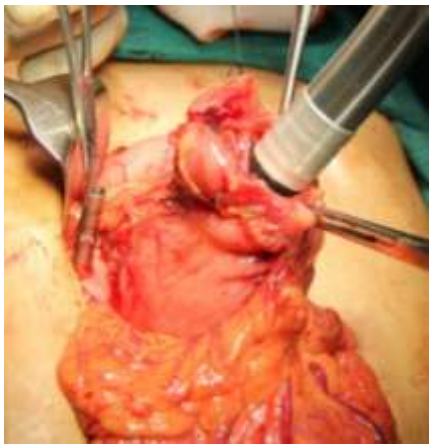
ARCADE PRESERVATION

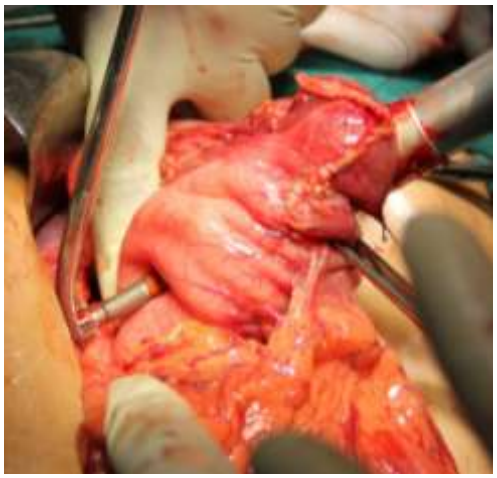




TOTALLY STAPLED BILROTH I PROCEDURE







Antrectomy without arcade preserving was done in 8 patients.Reconstruction was done by

Bilroth I -14 patients

Bilroth II -6 patients

We had one type II A Corrosive gastric stricture patient. He was managed by stricturoplasty. Four patients with type II B Corrosive gastric stricture patients were treated by Pylorus Preserving Antrectomy.

We had three type III corrosive gastric stricture patients. These patients were treated by circumferential sleeve resection and gastro gastrostomy. Five patients with type IV corrosive gastric stricture were managed by total gastrectomy. Reconstruction of these five patients was done by Roux-en-Y technique.

One patient with type V gastric stricture was managed by limited esophagogastrectomy. Intraoperative endoscopy was needed in this patient to define the extent of gastroesophageal junctional stricture.

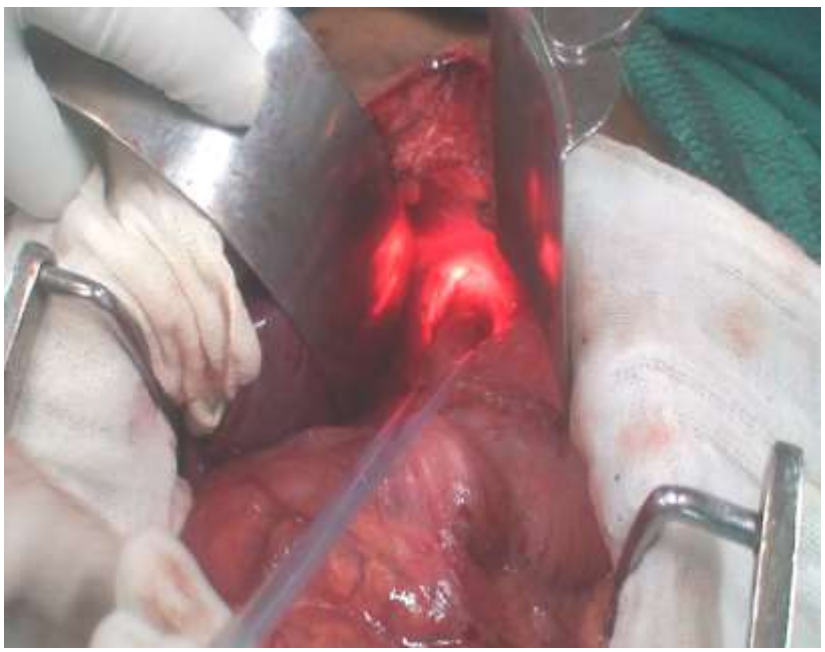
Three patients with type VI gastric stricture were managed by Gastrojejunostomy. Since these patients had first part of duodenal involvement, they were treated by bypass procedure rather than resection.

BARIUM MEAL OF THE PATIENT WITH TYPE III STRICTURE

GASTRO GASTROSTOMY FOR TYPE III GASTRIC STRICTURE

BARIUM MEAL- TYPE IV GASTRIC STRICTURE

GE JUNCTION STRICTURE (TYPE V) INTRA OPERATIVE ENDOSCOPY GUIDANCE



BARIUM MEAL - TYPE V GASTRIC STRICTURE

We had 11 patients with type VII Corrosive gastric stricture. These strictures are otherwise called as GASTROCELE [55].

They presented with

Dysphagia	11
Dull abdominal pain	3
Acute abdominal pain and respiratory embarrassment	2
No abdominal symptoms	6

Majority of patients who were suspected to have Gastrocele by the clinical examination underwent Barium meal and CECT Abdomen with oral contrast to confirm the clinical findings.

The management of these patients was dependent on the length of oesophageal stricture and the extent of gastric stricture. Patients with short oesophageal stricture were managed by retrograde dilatation. Patients with long oesophageal stricture needed coloplasty. Those patients with stricture confined to the antrum were treated by antrectomy. Stricture extending into duodenum were managed by Gastrojejunostomy.

11 patients with type VII Gastric stricture in our series were managed by

Antrectomy, Coloplasty - 5

Gastrojejunostomy, Coloplasty - 3

Antrectomy, Retrograde dilatation – 2

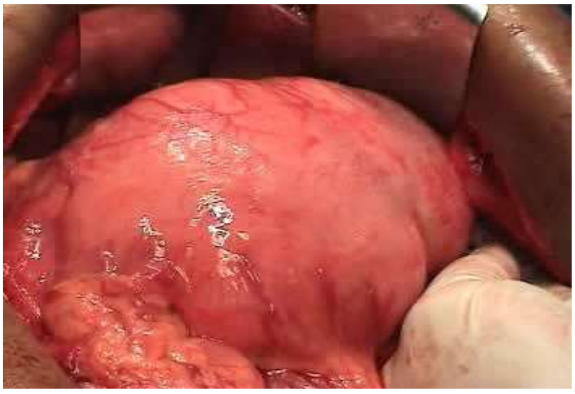
One patient expired during preoperative period due to aspiration and cardiorespiratory arrest.

Patients who were on feeding jejunostomy, it was retained .For other patients, feeding jejunostomy was done for nutritional support since almost all the patients were undernourished prior to surgery.

Endoscopic dilation as a primary modality was done for associated oesophageal stricture in 11out of 20 patients in our series.

Barium meal – Gastrocele

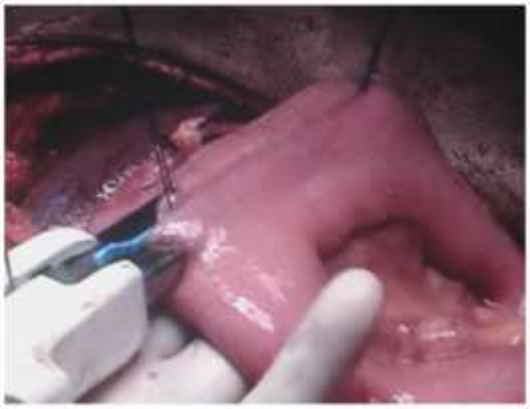
Intraoperative - Gastrocele



Gastrocele decompression

Antrectomy

Gastrojejunostomy



Gastroduodenostomy

Pink colon over the chest

Post operative picture

Post operative period

Patients who underwent reconstructive procedures were managed at intensive surgical care unit in immediate post operative period. Elective ventilation was done for select patients who underwent coloplasty.Extubation was planned thereafter depending on patients general condition in combination with anaesthesiologist.

Intensive monitoring was done for all patients with basic blood investigations.Electrolyte abnormalities were corrected.All patients had chest physiotherapy. Jejunostomy feeding was started routinely on the first post operative period except those patients who underwent coloplasty.For coloplasty patients,feeding jejunostomy was started on the fourth post operative day

Complications

Out of 48 patients, one patient died in the preoperative period (type VII stricture) due to aspiration and cardiorespiratory arrest and another patient on 5 th post operative day (type I stricture) due to resistant hypokalemia.

Abdominal wound infection was noted in 10 (20.8%) patients. Wound infection was nil in patients who underwent totally stapled bilroth I anastamosis for type I corrosive gastric stricture.Among 8 patients who

underwent coloplasty along with antrectomy or gastrojejunostomy, three of them had neck leak.It was treated conservatively.

9 patients developed respiratory tract infection (18.75%) in the post operative period.These patients were managed by aggressive chest physiotherapy,antibiotics and bronchodilators.

One patient needed re-exploration for intra abdominal hematoma on the 10 th post operative day.Hematoma was evacuated and peritoneal lavage was done. Patient was stable after the procedure.

Hospital stay varied between 9 days to 24 days.Length of hospital stay was longer in patients who underwent coloplasty along with antrectomy compared to other procedures.

Among 3 patients who had neck leak, two patients developed anastamotic stricture at colopharyngeal anastamosis.They were treated with endoscopic dilatation and are on regular follow up.

DISCUSSION

Consumption of caustics is one of the common modes of poisoning in developing countries.It is the common cause of benign stricture of upper gastro intestinal tract [57]. Acid ingestion is more common than alkali ingestion in India as concentrated acids are much cheaper than alkaline toilet cleaner [58]. Most of the our patients have consumed toilet cleaning acid.

Either acid or alkali produces reflex pyloric spasm leading to pooling of acid leading to antral stenosis, hourglass type of deformity or linitis plastica[59]. Zargar et all descibed that acute gastric injury was present in 85.4% of their patients who had consumed acid involving mainly the distal stomach and 44% had late complications like pyloric or antral stricture and linitis platica. He also stated that duodenum is relatively spared because of pyloric spasm produced by irritant acid in the antrum and the alkaline pH of the duodenum.

Since ours is a tertiary center and also center for excellence in upper GI surgery,we receive cases from all over the state and sometimes from other states. We had 48 patients with corrosive gastric stricture in thirty months.To best of our knowledge ,this is the largest reported case series in the literature about corrosive gastric stricture in the shorter duration of study period.

Ananthakrishnan et all has published their experience with Chronic Corrosive Injuries of the Stomach— 109 Patients Over Thirty Years [60].

V.K.Kapoor from SGPGI insititute from India has also published their experience with 28 patients over a period of 15 years [61].

Most of the our patients were young female in the second or third decades.Most of the them ingested toilet cleaning acid (83.3%). Majority of patients ingeted acid with sucidal intention (87.5%).Duration between corrosive ingestion and the development of stricture varied from 2 months to 35 months.

Most of the patients presented with vomiting (66.6%) , dysphagia (43.75%), respiratory tract infection (41.66%) and abdominal pain (31.25%). All patients had weight loss (100%). Among 48 patients, 13 patients (27.08%) had hyponatremia (sodium <130). 9 patients with type I stricture, 3 patients with type VII stricture and 1 patient with type IV had hyponatremia. 15 patients (31.25%) had lower potassium level at the time of admission.8 patients with type I stricture, 3 with type VII, 1 each with type II a, type II b, type III, type IV had hypokalaemia. Hyponatremia and Hypokalaemia were corrected prior to surgery.10 patients (20.83%) had hypoalbumnemia (albumin <3.5).

Upper GI scopy and barium meal was done for almost all of these patients to locate the level of the stricture.28 patients in our series had

stricture isolated to the stomach. Remainig 20 patients had associated oesophageal stricture.

Barium enema and colonoscopy were done only in patients with combined oesophageal and gastric stricture planned for coloplasty (type VII). Mesentric angiography was not done in those patients.

We have classified corrosive gastric stricture into seven types based on the location and extent of strictures

Type I - Stricture < 5 cm from pyloro duodenal ring

Type II - Stricture >5 cm from pyloro duodenal ring

Type II A – Stricture < 1 cm

Type II B – Stricture > 1 cm

Type III – Midbody stricture

Type IV – Diffuse stricture

Type V – Proximal or Gastro-oesophageal junctional stricture

Type VI –Distal gastric stricture extending into duodenum

Type VII – Double stricture involving distal oesophagus and distal stomach

Among seven types, corrosive antral stricture (Type I) was the commonest in our sreies (41.66%). Combined lower oesophageal and lower gastric stricture (Type VII) was the second most common type (22.91%). Type II stricture was in 10.41%, Type III stricture was in 6.25%, Type IV stricture was in 10.41%, Type V was in 2.08% and Type VI was in 10.41%.

Ananthakrishnan et all has classified corrosive gastric stricture into five types[22]. But their classification did not include double gastric stricture and also the proximal stricture involving Gastro-oesophageal junction.

Although endoscopic dilatation has a role in managing gastric strictures particularly for type I, we routinely do resectional procedure rather than endoscopic dilatation for corrosive gastric strictures. Endoscopic dilation as a primary modality was done for associated esophageal stricture in 11 patients.

Optimal timing and type of surgery for gastric stricture are still unclear[62]. But Hwang et all proposed early definitive operation to manage these injuries[63]. In corrosive stricture of stomach,surgery is tailored according to the extent of gastric involvement and presence of associated esophageal strictures[42]. We do surgery only for established stricture and symptomatic patients.

Type I stricture in our sreies was managed by arcade preserving antrectomy in 12 patients and antrectomy without arcade preserving in 8 patients. Reconstruction was done by Bilroth I in 14 patients and Bilroth II in remaining 6 patients.

Arcade preserving antrectomy has the advantage of preserving both right gastric vessels and right gastro epiploic vessels.The remanant stomach can be used for gastric pull up procedure if needed.

One patient with type II A stricture was managed by stricturoplasty.Four patients with type II B stricture were treated by pylorus preserving antrectomy.

Three patients with type III gastric stricture were managed by circumferential sleeve resection and gastrgastrostomy.Total gastrectomy was done for 5 patients with type IV stricture.Reconstruction was done with Roux-en-Y jejunal loop.

One patient with type V gastric stricture was managed by limited esophagogastrectomy.Three patients with type VI gastric stricture were managed by Gastrojejunostomy.

Patients with combined stricture of short segment esophageal stricture and antral stricture can be managed by antrectomy, retrograde dilatation of oesophageal stricture and follow up for esophageal stricture. Long segment, undilatable or multiple oesophgeal stricture with antral

stricture are managed with antrectomy with coloplasty (Oesophago or pharyngocolic, cologastric, ileo colic anastamosis). Patients with long segment, undilatable or multiple oesophgeal stricture with coexisting distal stricture extending into the duodenum are managed by gastrojejunostomy and coloplasty. Combined long osophageal stricture and diffuse deformity of stomach can be managed with leaving the diseased stomach and bypassing the upper gastrointestinal tract with coloplasty and colojejunal anastamosis.

We had 11 patients with combined esophageal and distal gastric stricture.It was managed by Antrectomy with Coloplasty in 5 patients,Gastrojejunostomy with Coloplasty in 3 patients and Antrectomy with Retrograde dilatation in 2 patients. One patient expired during preoperative period due to aspiration and cardiorespiratory arrest.

Patients who were on feeding jejunostomy,it was retained. For other patients, feeding jejunostomy was done for nutritional purposes since most of the patients were undernourished prior to surgery.

Out of 48 patients, one patient died during preoperative period and another on the 5 th post operative day due to resistant hypokalemia. Abdominal wound infection was noted in 10 (20.8%) patients. Out of 8 patients who underwent coloplasty, 3 patients had neck leak which settled with consevative line of management. Respiratory tract infection was

noted in 18.75% patients. One patient needed re-exploration for intraabdominal hematoma on the 10 th post operative day.

Hospital stay varied between 9 days to 24 days. Length of hospital stay was longer in patients who underwent coloplasty along with antrectomy compared to the other procedures.

All patients had psychotherpy and psychatric counselling. Among 48 patients, 8 patients had mild to moderate depressive illness. They were managed with appropriate medications.Remaining patients were found to be physchologically normal on evaluation. We have the reasons to believe that they suffered from either sub clinical physhatric disorder or the ingestion was an impulsive reaction to the emoitional trauma.

All patients are being followed at regular intervals in the outpatient department by the way of history of change in the grade of dysphagia,weight gain,return to job, and clinical examination and their QOL is being analysed.

Endoscopy was routinely done in 3 and 6 months post operatively for all of these patients.

Two of three patients who developed neck leak had anastamotic stricture at the level of oesophagocolic anastamosis. They were managed by endoscopic dilatation. No patients with gastroduodenal, gastrojejunal, gastrogastric and oesophagojejunal anastmosis had anastomotic stricture.

CONCLUSION

Our study on corrosive gastric stricture have certain implications to suggest :

Corrosive stricture can be isolated to stomach or associated with oesophageal stricture.

The morbidity and mortality of gastric corrosive stricture can be significantly reduced by adequate preoperative nutritional optimisation and pre operative evaluation to asses the extent of injury.

This new working Classification will help in deciding the surgical strategy to be adopted in the management of corrosive gastric stricture according to the site of the stricture.

The long-term results, particularly in the relief of symptoms,weight gain and quality of life are better after surgical management of corrosive gastric stricture.

BIBLIOGRAPHY

1. Winkel R et al necrosis of the oesophagus. Ugeskrift forlaeger. 2009

2. Zwischenberger JB et all Esophageal Diseases. Am J Respir Crit Care Med.2002

3. Kikendall J N. Caustic injuries. Gastroenterol North American Clinics. 1991

4. Kakar AK et al (1988) Cicatricial stenosis of stomach by corrosive ingestion. Aust NZ J Surgery

5. Singer HA (1930) et all, Gastric sequelae of corrosive poisoning. Arch Intern Med 40

6. Christesen H. Ingestion of corrosive agents Ugeskrift for laeger. 1993. Aviloglu K et al. The results of caustic ingestions. Hepatogastroenterology. 2004

7. Ramasamy K et all. Ingestion of Corrosive in adults. Journal of clinical gastroenterology. 2003

8. Mehta SK et all. Ingestion of corrosive acids. Injury to upper gastrointestinal tract and natural history. Gastroenterology. 1989

9. Ogunleye et all,Chemical injuries of the oesophagus: aetiopathological issues in Nigeria,J Cardiothorac Surgery. 2009

10. Bajor J,et all Long-term results of balloon dilation for benign gastric outlet stenosis.Endoscopy 2003

11. Wig JD et all. Endoscopic balloon dilatation for benign gastric outlet obstruction. J Gastroenterology Hepatol 2004

12. Lee SD et all. patients with benign gastric outlet obstruction ,need of surgery after endoscopic balloon dilation. Am JGastroenterol 1996

13. Di ZHet al. Gastric outlet obstruction , benign anastomotic stricture:treatment by fluoroscopically guided balloon dilation. J Vasc Interv Radioliogy 2005

14. Gross KR et all. Successful dilatation of pyloric stricture resistant to balloon dilatation with electrocautery using a sphincterotome.J Clin Gastroenteroogy 1996

15. Yamagishi H et all. Combined use of electrosurgical incisions and balloon dilatation for the treatment of refractory postoperative pyloric stenosis. Gastrointest Endoscopy 2001

16. Singh K et all.Intralesional steroid injections for caustic induced pyloric stenosis. Endoscopy 1998

17. K. Ragunath et all, "Esophageal stents for benign refractory strictures: a meta-analysis," Endoscopy, vol. 43, pp. 386–393, 2011

18. V.K.Kapoor surgical management of corrosive stricture of stomach.IndianJ.of Gastroenterology,2004

19. M Vadivelan et al, Indian Journal of Clinical Practice, Vol. 23, No. 3, August 2012

20. Lahoti Corrosive injury to upper gastrointestinal tract.Indian Jornal of gastroenterology 1993.

21. Ingestion of Caustic Substances in Adults: A Review Article Zanina Peresk, et allindian journal of toxicology 2013

22. Corrosive Injuries of the Stomach— Experience of 109 Patients Over Thirty Years Ananthakrishnan, G. Parthasarathy et all World j surg 2010

23. Critical Care Toxicology: Diagnosis and Management of the Critically Poisoned Patient. 1st edition, Brent, Donovan (Eds.) 2005

24,25. Caustics. Goldfranks Toxicologic Emergencies. 8th edition

26. Fordtrans Gastrointestinal and Liver Disease: Pathophysiology /Diagnosis /Management. 8th edition, Lawrence, Lawrence J. Brandt (Eds.) 2006

27. Squamous Carcinoma of the Stomach Following Corrosive Acid Burns Henry Eaton et all, British Journal of Surgery pages 382–387, May 1972

28. Korolev MP et all. Treatment of patients with combined burn strictures of the esophagus and stomach.Vestn Khir Im I I Grek. 2005

29. Post-corrosive injuries of upper gastrointestinal tract Chibishev A**.** Biol. Med. Sci., XXXI/1 (2010), 297–316

30. De Mello Filho F et all. Treatment of caustic ingestion: an analysis of 239 cases. Diseases of the Esophagus.2002

31. Assens P et all. (2005): Menagement of caustic ingestion in adults, Volume 74, Issue 2, Pages 146–1488 Dec**.**

32. Lin YD et all 2005: Menagement of corrsive esophageal burns in 149 cases, J Thorac cardiovascular Surg, Aug; 130(2): 449–55

33. Poley JW et al. Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy. Gastrointest Endosc 2004

34. Cummins GE et al (2003) Application of the Meckel's scan in a case of gastric corrosive injury. Pediatr Surg In

35. Kikendal JW. 1991: Caustic ingestion injuries. Gastroenterol Clin North Am.; 20(4): 847–857

36. Ren-Jie Yang et all 2003: Restenosis following balloon dilatation of benign esophageal stenosis, World J Gastroenterol November;9(11): 2605–2608

37. Ray JD et al (1998) Intralesional steroid injections for caustic induced pyloric stenosis. Endoscopy30:734–736

38. Lucas CE. Et all 2008: management of caustic injury in the upper gastrointestinal tract in 95 adult patients in an urban medical center.

39. Alinejad A. (2000): Caustic injury to upper gastrointestinal tract, Shiraz university of medical sciences

40. Katzka A. et all (2001): Caustic Injury to the Esophagus Current Treatment Options in Gastroenterology;

41 Bower RH. (1998): complications of total parenteral nutrition.; J Parenteral Enteral Nutr;

42. Sikora SS et all. (2004): Surgical management of corrosive strictures of stomach, Jndian J Gastroenterol; Sep-Oct: 23(5): 178–80.

43. M, Horva′th OP. Long-term results of balloon catheter dilation for benign gastric outlet stenosis.Endoscopy 2003;35:490-495

44. Wig JD et all. Endoscopic balloon dilatation of benign gastric outlet obstruction. J Gastroenterology Hepatol 2004

45. Nagi B, et al. Endoscopic balloon dilation in caustic-induced chronic gastric outlet obstruction. Gastrointest Endosc 2009

46. Kim TH, et al. Gastric outlet obstruction by benign anastomotic stricture: treatment by fluoroscopically guided balloon dilation. J Vasc Interv Radiol 2005;

47. Singh P et all. follow-up of patients with gastric outlet obstruction due to peptic ulcer disease treated with endoscopic balloon dilatation and drug therapy. Gastrointest Endosc 2007

48. Singh K et all.Intralesional steroid injections for corrosive induced pyloric stenosis. Endoscopy 1998

49. Surgical management of corrosive strictures of stomach shaleen agarwl Indian journal of gastroenterol 2004

50. Dhar P et al (1996) Elective surgery for corrosive-induced gastric injury. World J Surg 20:703–706

51. Chen MF et all (1996) Surgical treatment of gastric outlet obstruction after corrosive injury—can early definitive operation be used instead of staged operation? Int Surg 81:119–121

52. Eaton H.Squamous cell Ca stomach following corrosive acid burns Br.J.S 1972

53. Surgical management of corrosive strictures of stomach shaleen agarwl Indian journal of gastroenterol 2004

54. Sharma R et al (2003) Corrosive-induced gastric outlet obstruction. Yonsei Med J 44:991–994

55. Chandramohan SM. complication of combined oesophageal and antral corrosive strictures. Interact Cardiovasc Thorac Surg. 2009

56. Radjendiran P et al (1991) Delayed gastric outlet obstruction after esophagocoloplasty— clinical presentation with massive megacolon. Indian J Thorac Cardiovasc Surg 7:99

57. Zargar SA et al ingestion of strong corrosive alkalis.Am J Gastroenterol 1992

58. Mehta S et al Spectrum of injury to upper gastrointestinal tract and natural history. Gastroenterology. 1989

59. Roy M Jr ,N Eng J med 1962

60. Ananthakrishnan Chronic Corrosive Injuries of the Stomach—A Single Unit Experience of 109 Patients Over Thirty Years World J Surg (2010)

61. V.K. Kapoor(2004): Surgical management of corrosive strictures of stomach, Jndian J Gastroenterol; Sep-Oct: 23(5): 178–80

62. Adarsh Choudry et all, elective surgery for corrosive induced gastric injury, World journal of surgry,vol.20,nov 6 July 1996

63. Hwang, Chen MF (1996) treatment of gastric outlet obstruction after corrosive injury—can early definitive operation be used instead of staged operation? Int Surg 81

CASE REPORT FORM

Name -

Age -

Sex -

Address -

Occupation (level) -

Consumption of acid/alkali

Intention for consumption

H/o difficulty in swallowing

H/o cough with expectoration

H/o difficulty in breathing

H/o abdominal pain

H/o abdominal distension

H/o vomiting

H/o fever

H/o alcohol intake- amount, frequency

H/o comorbid illness

DM,SHT,BA,TB,IHD

General examination

Examination of oral cavity

Respiratory system examination

Cardiovascular system examination

Abdominal examination

Investigations

Complete blood count

Serum electrolytes

Blood sugar

Renal function tests

Urea

Serum creatinine

Liver function tests

Bilirubin

SGOT

SGPT

SAP

Serum albumin

Prothrombin time

ECG

Chest X-ray

X-ray abdomen

Ultrasonagram abdomen

Barium swallow

Upper GI endoscopy

ENT examination

Psychiatric opinion

Type of surgical Procedure

Outcome

Wound infection

Respiratory infection

Anastomotic leak

In hospital stay

In hospital mortality

INFORMATION TO PARTICIPANTS

Title: **"Analysis of presentation, management and outcome of corrosive gastric strictures"**
Principal Investigator:
Co-Investigator(if any):

Name of Participant:

Site :
You are invited to take part in this research/ study/procedures/tests. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

What is the purpose of research?
Corrosive gastric strictures is an uncommon disorder characterized by vomiting. It usually presents as inability to take food, abdominal pain & vomiting . These symptoms may last for _____(usual course of the disease). We want to test the efficacy and safety of a new _____ (drug / intervention / surgery /procedure/lab test) in this disease/condition.

We have obtained permission from the Institutional Ethics Committee.

The study design
Retrospective study

Study Procedures
The study involves evaluation of gastric stricture for which we will need barium swallow & upper gastrointestinal scopy .The planned scheduled visits involve visits at _____,_____,____, and_____(days/ weeks) after your initial visit. You will be required to visit the hospital _____ number of times during the study.

In addition, if you notice any physical or mental change(s), you must contact the persons listed at the end of the document.

You may have to come to the hospital (study site) for examination and investigations apart from your scheduled visits, if required.

The results of the research may provide benefits to the society in terms of advancement of medical knowledge and/or therapeutic benefit to future patients.

Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel, sponsors, Institutional Ethics Committee and any person or agency required by law like the Drug Controller General of India to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not loose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to stopping the treatment/discontinuing of procedures etc.

Signature of Investigator Signature of Participant

Date Date

INFORMED CONSENT FORM

Title of the study -**"Analysis of presentation, management and outcome of corrosive gastric strictures"**

Name of the participant: _____

Name of the Principal/Co-Investigator: _____

Name of the Institution: Department of surgical gastroenterology, Madras Medical College and Rajiv Gandhi government general hospital, Chennai

I,_____(name of participant), have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in "**Analysis of presentation, management and outcome of corrosive gastric strictures"**

(1) I have read and understood this consent form and the information provided to me.

(2) I have had the consent document explained to me.

(3) I have been explained about the nature of the study.

(4) I have been explained about my rights and responsibilities by the investigator.

(5) I have informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatments.

(6) I have been advised about the risks associated with my participation in the study.

(7) I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.

(8) I have not participated in any research study within the past _____ month(s).

(9) [I have not donated blood within the past _____months

(10) I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in the hospital

(11) I am also aware that the investigators may terminate my participation in the study at any time, for any reason, without my consent.

(12) I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Government agencies, and ethics committee. I understand that they may inspect my original records.

(13) I understand that my identity will be kept confidential if my data are publicly presented.

(14) I have had my questions answered to my satisfaction.
(15) I consent voluntarily to participate as a participant in the research study.

I am aware, that if I have any questions during this study, I should contact the investigators. By signing this consent from, I attest that the information given in this document has been clearly explained to me and understood by me. I will be given a copy of this consent document.

For adult participants

Name and signature / thumb impression of the participant (or legal representative if participant incompetent):

(Name) _____(Signature)_____ Date: _____

Name and signature of impartial witness (required for illiterate patients):

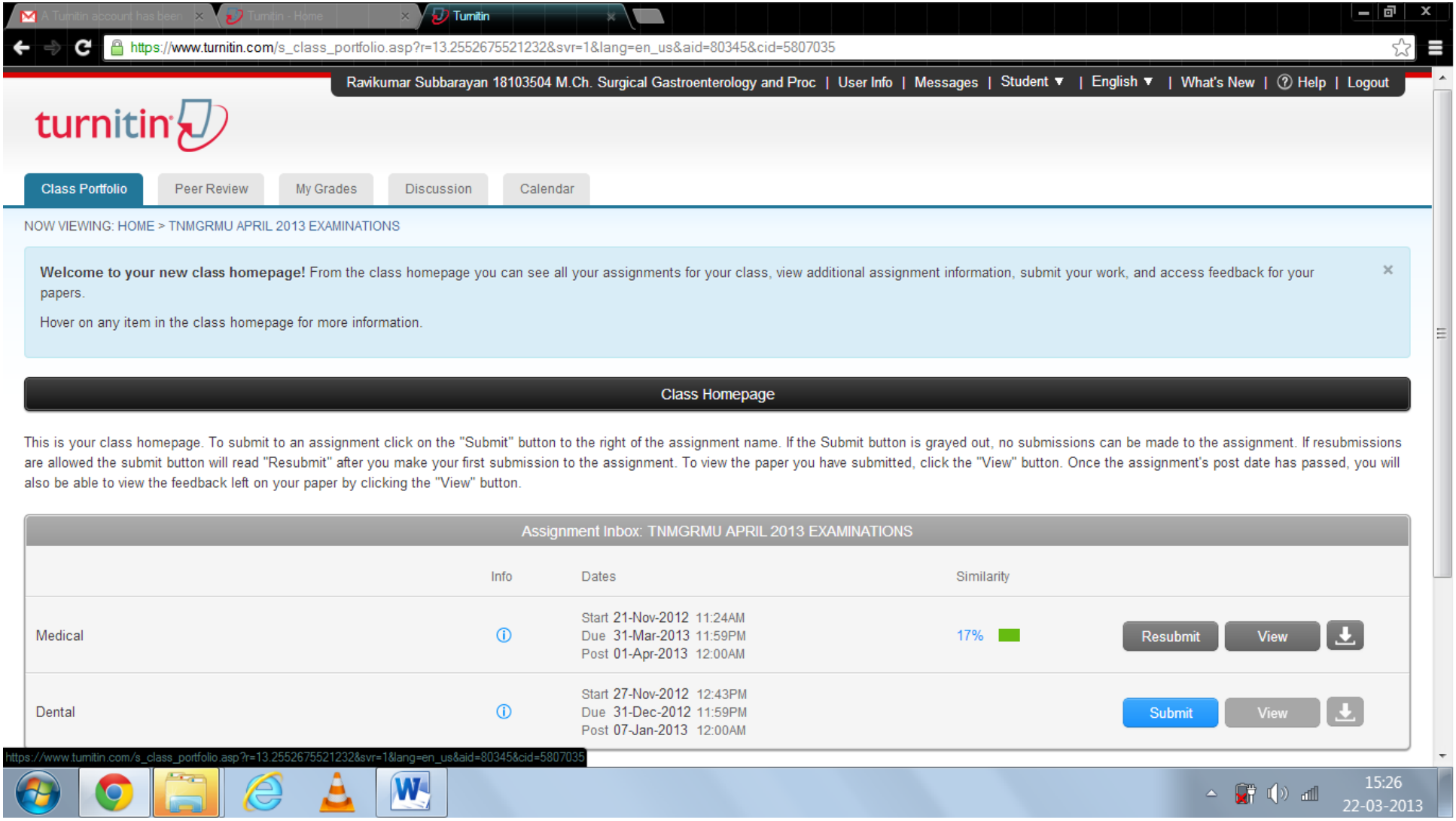
(Name) _____ (Signature)_____ Date:_____

Address and contact number of the impartial witness:

_____

Name and signature of the Investigator or his representative obtaining consent:

(Name)_____(Signature)_____ Date)_____


A Turnitin account has been
Turnitin - Home
Turnitin
https://www.turnitin.com/s_class_portfolio.asp?r=13.2552675521232&svr=1&lang=en_us&aid=80345&cid=5807035
Ravikumar Subbarayan 18103504 M.Ch. Surgical Gastroenterology and Proc | User Info | Messages | Student | English | What's New | Help | Logout
turnitin
Class Portfolio
Peer Review
My Grades
Discussion
Calendar
NOW VIEWING: HOME > TNMGRMU APRIL 2013 EXAMINATIONS
Welcome to your new class homepage! From the class homepage you can see all your assignments for your class, view additional assignment information, submit your work, and access feedback for your papers.
Hover on any item in the class homepage for more information.
Class Homepage
This is your class homepage. To submit to an assignment click on the "Submit" button to the right of the assignment name. If the Submit button is grayed out, no submissions can be made to the assignment. If resubmissions are allowed the submit button will read "Resubmit" after you make your first submission to the assignment. To view the paper you have submitted, click the "View" button. Once the assignment's post date has passed, you will also be able to view the feedback left on your paper by clicking the "View" button.
Assignment Inbox: TNMGRMU APRIL 2013 EXAMINATIONS
Info
Dates
Similarity
Medical
Start 21-Nov-2012 11:24AM
Due 31-Mar-2013 11:59PM
Post 01-Apr-2013 12:00AM
17%
Resubmit
View
Dental
Start 27-Nov-2012 12:43PM
Due 31-Dec-2012 11:59PM
Post 07-Jan-2013 12:00AM
Submit
View
https://www.turnitin.com/s_class_portfolio.asp?r=13.2552675521232&svr=1&lang=en_us&aid=80345&cid=5807035
15:26
22-03-2013

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To

Dr.S.Ravikumar,
III Year P.G, M.Ch Surgical Gastro Enterology,
Department of Surgical Gastroenterology,
Madras Medical College & RGGGH Chennai -3

Dear Dr.S.Ravikumar,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Analysis of presentation, management and outcome of corrosive gastric strictures" No.11022013.

The following members of Ethics Committee were present in the meeting held on 05.02.2013 conducted at Madras Medical College, Chennai -3.

1.	Dr.SivaKumar, MS FICS FAIS	--- Chairperson
2.	Prof. R. Nandhini MD Director, Instt. of Pharmacology ,MMC, Ch-3	-- Member Secretary
3.	Prof. Shyamraj MD Director i/c , Instt. of Biochemistry , MMC, Ch-3	-- Member
4.	Prof. P. Karkuzhali. MD Prof., Instt. of Pathology, MMC, Ch-3	-- Member
5.	Prof. A. Radhakrishnan MD Prof of Internal Medicine, MMC, Ch-3	-- Member
6.	Prof. S. Deivanayagam MS Prof of Surgery, MMC, Ch-3	-- Member
7.	Thiru. S. Govindsamy. BABL	-- Lawyer
8.	Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nadini 22/2/13
Member Secretary, Ethics Committee